In the Claims

Please amend claims **33** and **39** as follows:

Claim 33 (Amended Four Times) A method for directly identifying a non-endogenous candidate compound as a compound having activity selected from the group consisting of inverse agonist activity and agonist activity to an endogenous G protein coupled cell surface receptor, wherein a location of expression of said receptor has been identified from a mammalian tissue source and has been correlated with at least one physiological function in a mammal, comprising the steps of:

(a) selecting an endogenous G protein coupled cell surface receptor, wherein the endogenous ligand for said receptor has not been identified;

(b) determining the location of expression of said receptor in a mammalian tissue source and correlating the expression location of said receptor with at least one mammalian physiological function of interest, wherein said location and said correlated physiological function are selected from the group consisting essentially of:

receptor not known

Location:	Correlated Physiological Function:
116. ventromedial hypothalamus	116. food intake

(c) subjecting said receptor to constitutive receptor activation to establish a non-endogenous constitutively activated G protein coupled cell surface receptor;

(d) contacting a non-endogenous candidate compound with said non-endogenous constitutively activated G protein coupled cell surface receptor of step (c);

(e) determining, by measurement of the compound efficacy at said contacted receptor, whether said non-endogenous candidate compound has inverse agonist activity or agonist activity to said receptor of step (c); and

(f) directly identifying a non-endogenous candidate compound of step (e) having inverse agonist activity as an inverse agonist to said receptor of step (c), or having agonist activity as an agonist to said receptor of step (c);

(g) selecting an inverse agonist to reduce a selected physiological function of step (b) correlated with the tissue-expression location for said receptor of step (a), or selecting an agonist to enhance a selected physiological function of step (b) correlated with the tissue-expression location for said receptor of step (a); and

(h) contacting said inverse agonist with a mammal comprising said receptor of step (a) and confirming that said inverse agonist reduces said selected physiological function, or contacting said agonist with a mammal comprising said receptor of step (a) and confirming that said agonist enhances said selected physiological function

wherein said directly identified non-endogenous candidate compound of step (f) was not, prior to such direct identification, indirectly identified as an agonist or antagonist to said endogenous G protein coupled cell surface receptor.

Claim 39 (Amended Four Times) A method for directly identifying a non-endogenous candidate compound as a compound having activity selected from the group consisting of inverse agonist activity and agonist activity to an endogenous constitutively activated G protein coupled cell surface receptor, wherein a location of expression of said receptor has been identified from a mammalian tissue source and has been correlated with at least one physiological function in a mammal, comprising the steps of:

(a) selecting an endogenous constitutively activated G protein coupled cell surface receptor, wherein the ligand for said receptor has not been identified;

(b) determining the location of expression of said receptor in a mammalian tissue source and correlating the expression location of said receptor with at least one mammalian physiological function of interest, wherein said location and said correlated physiological function are selected from group consisting essentially of:

Location:		Correlated Physiological Function:
116. ventromedial hypothalamus		116. food intake

(c) contacting a non-endogenous candidate compound with said endogenous constitutively activated G protein coupled cell surface receptor of step (a);

(d) determining, by measurement of the compound efficacy at said contacted receptor, whether said non-endogenous candidate compound has inverse agonist activity or agonist activity to said receptor of step (a); and

(e) directly identifying a non-endogenous candidate compound of step (d) having inverse agonist activity as an inverse agonist to said receptor of step (a), or having agonist activity as an agonist to said receptor of step (a);

(f) selecting an inverse agonist to reduce a selected physiological function of step (b) correlated with the tissue-expression location for said receptor of step (a), or selecting an agonist to enhance a selected physiological function of step (b) correlated with the tissue-expression location for said receptor of step (a); and

(g) contacting said inverse agonist with a mammal comprising said receptor of step (a) and confirming that said inverse agonist reduces said selected physiological function, or contacting said agonist with a mammal comprising said receptor of step (a) and confirming that said agonist enhances said selected physiological function

wherein said directly identified non-endogenous candidate compound of step (e) was not, prior to such direct identification, indirectly identified as an agonist or antagonist to said receptor.

Attached hereto is a marked-up version of the changes made to the claims by the current amendment. The attached page is captioned **"Version with markings to show changes made."**